Cancer as a mini-evolutionary process(International & Interdisciplinary Symposium on What is Evolution? Bicentennial of Charles Darwin's Birth)

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This switch also would bring diversity to some extent because useful mutations could be introduced. Needless to say, diploidization connotes fertilization, and haplodization connotes meiosis.

I also stress that this alternation cycle can easily accept the advanced form of sexual reproduction beginning with the haploid cell fusion. Because the genomic duplication (Du) is equivalent to the genomic cell fusion (Fu) as for diploidization, one of the Du-Di modules of the haploid cell cycle is easily replaced with a Fu-Di motif: it is nothing but the one-step meiosis. If a transition from diploid to haploid occurs in such the way of Fu-Du-Di-Di- instead of Du-Di-Di-, it is nothing but the two-step meiosis.

Taken altogether, the alternation of asexual cycles between diploid and haploid is considered the primitive form of sexual reproduction.

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Cancer as a mini-evolutionary process

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Cancers result from the accumulation of inherited and somatic mutations in oncogenes and tumor suppressor genes. Those mutations increase the net reproductive rate of cells. Many aspects of carcinogenesis can be handled as evolutionary processes.

[1] Chromosomal instability (CIN) is a feature of most human cancers. From the mathematical analysis of situations where inactivation of one or two tumor suppressor genes is required for tumorigenesis, we conclude that CIN is likely to emerge first and then enhance the risk of cancer.

[2] Most epithelial tissues have common architecture -- a tissue is organized into numerous small compartments, and within each compartment includes a few stem cells and numerous differentiated cells. This design can slow down delay the onset of cancer.

[3] The ABL tyrosine kinase inhibitor imatinib in chronic myeloid leukemia (CML) serves as a model for molecularly targeted therapy of cancer. We show that a four-compartment model can explain the kinetics of the molecular response to imatinib in a 169-patient data set. We also calculate the probability of developing imatinib resistance mutations.